Clinical Cases.

A CONTRIBUTION TO THE STUDY OF LANDRY'S ASCENDING PARALYSIS.*

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Before giving the clinical history of a case of acute ascending paralysis observed by me, it may not be out of place to remember that in 1859 Landry described a few cases of ascending paraplegia, without lesions in the spinal cord, and named this form of paraplegia "paralysie ascendante aigue," and Kussmaul in the same year reported two rapidly fatal cases of spinal paralysis, where the post-mortem appearance of the cord was found to be apparently normal.

The attempt of Petit fils to identify Landry's ascending paralysis with poliomyelitis anterior acuta or subacuta, *i. e.*, to look upon it as a variety of the latter disease, differing in degree only, has been made again quite recently by Prof. Immermann, more in the form of a suggestion, as I shall show later on. But in consideration of more recent facts bearing upon the subject, and particularly of the negative results of autopsies made by Westphal upon some clinically well-observed cases of Landry's paralysis, those attempts of Petit fils and others cannot be called successful. It is perhaps somewhat different with the theory recently put forth by Roger, who believes that many cases of the favorable forms of ascending paralysis are cases of polyneuritis acuta, of infectious or other origin, in which the spinal cord is either not at all or, when so, only secondarily affected.

In *L'Encéphale*, No. 2, 1885, G. H. Roger, published an interesting article, "Les Névrites Périphériques." He discourses on the clinical pictures which are produced by affections of the peripheral nerves, particularly in polyneuritis, with symptoms similar to those of medullar disease. He speaks first of localized neuritis both after trauma and infectious disease, which may produce

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trophic disturbances and paralysis, without any participation of the spinal cord. In eczema, pemphigus, herpes zoster, etc., the ganglionic bodies may remain intact, or may undergo secondary degeneration when the neuritis moves in a central direction. In cases of simultaneous peripheral and spinal affections, a correct diagnosis is of course difficult, and up to the present, says he, we have paid almost exclusive attention to the central lesions, although the course of the disease has in many cases shown that the lesion ascended from the periphery to the centre. He maintains that many cases of Landry's paralysis, the literature of which he gives in extenso, belong to the peripheral nervous affections, as well as Duchenne's "paralysie générale spinale subaigue," and "paralysie diffuse." Several cases reported by Leyden and Eisenlohr, in which they found, after ascending muscular atrophy and paralysis, the principal lesions in the peripheral nerve branches, Roger claims also as essentially peripheral neuritis.

The usual course of the disease is, according to Roger, the following: A brusque attack of severe pain in the extremities, generally the lower, often considerable fever, soon after paralytic condition, but no contractures; diminished or abolished tendon reflex, cutaneous reflex variable, the paralyzed muscles become rapidly atrophic, and the incipient hyperæsthesia soon gives place to anæsthesia. Pressure over the affected nerves is exquisitely painful, the electric irritability generally diminished, the intellect remains undisturbed. The result is mostly favorable, but complete or partial recovery takes place slowly. When death ensues, it occurs generally by asphyxia, under pseudo-bulbar symptoms. The lesions which may be found in such cases in the spinal cord, or its meninges, are only secondary, though they have often been looked upon as primary. The differential diagnosis between diffuse polyneuritis and diffuse myelitis is often very difficult, according to Roger. The absence of visceral disturbances furnishes the best argument for the assumption of peripheral neuritis.

As to the etiology of polyneuritis, we are still in the dark. Westphal lays much stress upon infection. Roger draws attention to the frequent coincidence of tuberculosis and polyneuritis. I do not know whether Erb has changed his views with regard to this point, but as late as 1879 he said that cases of progressive polyneuritis ought not to be mistaken for acute anterior poliomyelitis by the carefully observing practitioner, for the sharp pains and anæsthesias and paralyses limited to the affected nerves, and the rapid loss of their electric excitability, are sufficient to establish the differential diagnosis. It may be justifiable, according to these recent observations, to speak of two forms of acute ascending paralysis, the well-characterized spinal and less well-known peripheral. Evidently more clinical and anatomical research is required in order to arrive at a satisfactory understanding of the disease.

Paralysis ascendens acuta is clinically characterized by a motory paralysis, which, generally beginning at the lower extremities, af-

fects pretty rapidly the trunk and upper extremities, pretty often, also, the medulla oblongata ; the sensory nerves, bladder, and rectum are but little affected. The paralyzed muscles do not waste much, and do not change or lose their electric excitability. More or less fever may accompany the disorder, which pretty often terminates fatally by asphyxia, etc. At the autopsy no lesions are found in the cord.

Men are taken with Landry's paralysis more frequently than women ; most cases occur between the twentieth and fortieth years, but it has been observed later in life ; of hereditary or neuropathic influences nothing is known. Among the exciting causes : severe cold, acute febrile and infectious diseases, such as typhoid, diphtheria, variola, etc. Suddenly suppressed menses have been noted. Dr. Bablou and others have seen it after "coite dans la station." The influence of syphilis is doubtful. Landry, Hayem, and of late Westphal, Roger, Strümpell, and others, think that infection, of an unknown character as yet, may be the real and determining cause of ascending paralysis. In following the thought of infection, as a cause, the fact is important that in some of the carefully made autopsies changes similar to those in other infectious diseases have been seen in the liver, spleen, lymphatic glands, and intestinal follicles.

The salient features in the symptomatology of ascending paralysis are : In most cases slight fever, pain in the back and limbs, and great weakness, and various paræsthesias as premonitory symptoms, lasting from one or more days to two or three weeks ; paresis of lower limbs, then of the body, then of the upper limbs, disturbance of respiration through paresis of respiratory muscles. No objective signs of sensory paralysis, no ataxia, no atrophy of paralyzed muscles, as in poliomyelitis anterior acuta, though more or less emaciation may take place in the course of time. The electric excitability of the paralyzed muscles and nerves remains perfectly normal, and that is thus far a most important point in distinguishing the disease from central myelitis and poliomyelitis anterior subacuta. Of vaso-motor disturbances, erythema and profuse sweating have been noted, but no trophic cutaneous changes. Bed-sores have not occurred in a single case. In the majority of cases the tendon reflex is present in the beginning of the disease, but becomes diminished and abolished later on ; but it does not disappear so rapidly and completely as in poliomyelitis anterior acuta. The reports about the cutaneous reflexes vary considerably. Although the bowels are frequently constipated, and there may be occasional retention of urine, bladder and rectum are generally normal, and are never paralyzed, as in central myelitis.

The cerebral functions remain intact, also the cerebral nerves ; only the motor nerves in direct connection with the medulla oblongata may be affected sooner or later.

The paralysis progresses farther upwards in most cases, involving the medulla oblongata, and death occurs under the symptoms of bulbar paralysis. A fatal termination has been seen in

some cases within two or three days, and others again may have lasted two, three, or four weeks before they died. The average duration of the fatal cases seems to be from eight to twelve days. The cases which recover show a change for the better, generally within a few days of the duration of the severe symptoms. The improvement begins in the parts last affected, and progresses slowly, and many weeks may elapse before complete recovery ensues.

An opposite course of the paralysis, *i. e.*, descending from the upper part of the spinal cord, appears to be rare. In Ziemssen's Encyclopædia, edition of 1879, Erb says it must be considered doubtful as yet. My own case is such a one, and deserves to be placed on record for this, if for no other reason.

Some cases of poliomyelitis anterior subacuta may look exactly like Landry's paralysis for a while, and it may not be until the development of atrophy and the loss of electric reaction that we can establish the diagnosis of anatomical lesions of the spinal cord. Immermann's case, of which I shall speak hereafter, furnishes a striking example of the difficulty. A well-marked case of poliomyelitis anterior acuta is not progressive, does not attack the medulla oblongata, nor lead directly to a fatal termination ; the loss of faradic excitability and the muscular atrophy develop rapidly, and on these grounds mistakes in diagnosis might, therefore, be easily avoided. A doubt may arise in very mild cases. Again, in acute central and infectious myelitis we always have fever, severe sensory disturbances, early loss of all reflexes, sphincter paralysis, diminished electric excitability, bed-sores, and rapidly fatal termination.

There are certain forms of spinal syphilis which may present the clinical picture of acute ascending paralysis, and can be distinguished only by the patient's history and the positive results of specific treatment.

The prognosis of anterior ascending paralysis is always serious, and whether a case will terminate favorably or otherwise we can hardly say before some improvement has taken place.

In the treatment of this disease the antiphlogistic apparatus has been brought into action, with rather doubtful effects. Better results have been seen from the use of the wet pack, or warm baths with cold affusions, *i. e.*, procedures for stimulation of cutaneous action. Of remedies, good effects have been observed after giving the iodide of potassium and ergot. The application of a galvanic current of moderate strength upon the entire length of the spine is recommended by no less an authority than Erb. Good nutrition and careful stimulation of the patient are a matter of course.

On December 20, 1880, I was called to see Jacob H., living at College Point, L. I., æt. thirty-one, married, of apparently good constitution and average health, with no hereditary taint. He denies syphilis, and shows no lesions pointing to it. In his early life he often suffered from intermittent fever. At the age of twenty-one he began work in a barometer factory, and, as time went on, grew weak,

and had occasional slight fever and frequent vertigo, but no salivation or other symptoms of mercurial poisoning. He quit this work when he was 25, being compelled to do so by the continuation and increase of the above symptoms. He then lived in the country out West for four years, and returned to his home in good health at the age of twenty-nine.

About three weeks before I saw him, and as he was delivering meat to his customers in his open cart, on a wet and chilly morning, his horse ran away with him, and he had to put forth all his strength in trying to control his powerful animal. As he brought him to a stop he received no concussion nor other apparent injuries, but had a sick and tired feeling all the afternoon, and was hardly able to tend his shop. The day after he was about his place with difficulty, and on the third day he felt generally somewhat ill, had slight fever, and after some days of paræsthetic feelings, such as numbness, tingling, etc., creeping up from the fingers, he gradually lost the use of both arms. Two weeks later, and after similar paræsthesias of numbness, etc., etc., from the toes upwards, the legs became also paraplegic. No signs of paresis of sphincter muscles were noticed at that nor any subsequent time. His stools were retarded. The intercostal and other muscles of the trunk remained intact, nor had he any symptoms involving the medulla oblongata, but he felt some pain along the spine, and continued to have some pain and numbness here and there in his extremities. Up to the time of my visit the case had not been examined with any great care, but treated on general principles. I found the patient still unable to do more than slightly move his limbs while in bed; he could neither stand nor walk, and had not the power to grasp or hold any object with firmness. Cutaneous sensation appeared to be normal, but the patellar and Achilles tendon reflexes were absent. The limbs were not wasted, and the muscles responded to the electrical current. Pulse and temperature normal; urine also normal. The slight improvement in the patient's paralytic condition, noticeable to himself at the beginning of the fourth week, had not progressed any further.

Poliomyelitis, or ascending myelitis, could be excluded after this examination, and the case was diagnosed as one of Landry's ascending paralysis. A systematic course of galvanic treatment, massage, and warm baths appeared to be indicated to help the patient toward recovery. But it was impossible to carry this out at his home, and I had him, therefore, admitted to Roosevelt Hospital, where the late Dr. Evetzky became interested in the case. He agreed with me as to the great probability of its being a case of Landry's paralysis, with a comparatively good prognosis. He devoted a good deal of time and care to the man while at the hospital, treating him for over thirty days as above indicated. He was discharged cured, and was soon able to attend to his business again. I then lost sight of the patient, but had him looked up quite recently. He came to my office October 30th, of this year. He looked well enough, walked briskly and firmly, and said that

he was well able to attend to his business, but required a good deal of sleep, more so than five or six years ago. No lancinating pains, but occasional backaches; bowels somwhat constipated; urine 1,015, no abnormal ingredients; patellar tendon reflex totally absent on both sides; ability to stand on one foot with eyes closed not good; pupils quite normal; no sensory disturbances, no paræsthesias.

In the *Neurol. Centralbl.*, No. 9, 1885, Dr. Sorgenfrey reports a case of Landry's paralysis occurring in a robust man fifty-seven years of age, a week after getting his back wet in a cold drenching rain. After paralysis of all four extremities had developed, severe dyspnœa from paresis of respiratory muscles, and bulbar symptoms came on, and the patient was expected to die. However, as a *dernier ressort*, extract of ergot, gr. 1 every hour, was administered, until twenty grains had been taken, when the dangerous bulbar symptoms disappeared. The patient soon began to move his limbs again, and eventually recovered without any further medication. In the same number of the same journal, Dr. H. Mieth also reports a case of Landry's paralysis in a working man, aet. forty-two, whose recovery was greatly facilitated, he says, by the daily application of a mild descending current along the spine.

I bring a short notice of these two recent cases before you, because in them, as well as in those of Westphal and one of Hunnius and my own case, the early abolition of patellar and Achilles tendon reflex has been observed, the reappearance of which did not take place until after the complete recovery of the patient. It has not returned at all in my own patient. The vesical and rectal functions, cutaneous sensibility, and muscular electric excitability were found, and continued to be, normal in all these cases.

Prof. Immermann, in a paper read before this year's convention of German neurologists on poliomyelitis anterior acuta and Landry's paralysis, gave the history of a male patient, æt. twenty-two, who gradually developed paralysis of the upper and lower extremities of the abdominal muscles and bladder, while the sensory nerves remained intact. Immermann looked upon the case as one of ascending paralysis, but finding also that the patient had lost his tendon reflex, he for a while inclined more to the diagnosis of polyomyelitis anterior acuta. However, the electric reaction remaining normal, the bulbar symptoms disappearing, and the paralysis of the upper and lower extremities improving, and no muscular atrophy taking place, he returned to his former diagnosis of Landry's paralysis. The patient died of intercurrent pneumonia, and the autopsy showed recent inflammation in the anterior horns of the cervical, dorsal, and lumbar regions of the spinal cord. In concluding his remarks he throws out the suggestion that both clinical pictures may be due to one morbid process, the same in principle, but differing in degree.